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Office of Administrative Law Judges
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Issue Date: 17 May 2004

Case No. 2003-BLA-73

In the Matter of:
CHARLES BYRGE,
Claimant,

v.

PLATEAU MINING CORPORATION,
RAG AMERICAN COAL,
c/o ACCORDIA EMPLOYERS SERVICE
Employer,

and
DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

BEFORE: Administrative Law Judge Thomas F. Phalen, Jr.

APPEARANCES:
Jonathan Wilderman, Esq.
On behalf of Claimant

Scott White, Esq.
On behalf of Employer

DECISION AND ORDER – DENIAL OF BENEFITS

This is a decision and order arising out of a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977, 30 U.S.C. §§ 901-962, ("the Act") and the regulations thereunder, located in Title 20 of the Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title.[1]

[1] The Department of Labor amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended. These regulations became effective on January 19, 2001, and are found at 65 Fed. Reg. 80, 045-80,107 (2000)(to be codified at 20 C.F.R. Parts 718, 722, 725 and 726). On August 9, 2001, the United States District Court for the District of Columbia issued a Memorandum and Order upholding the validity of the new regulations. All citations to the regulations, unless otherwise noted, refer to the amended regulations.

On December 10, 2002, this case was referred to the Office of Administrative Law Judges by the Director, Office of Workers' Compensation Programs, for a hearing. (DX 93).[2] A formal hearing on this matter was conducted on July 17, 2003, in Price, Utah by the undersigned Administrative Law Judge. All parties were afforded the opportunity to call and to examine and cross examine witnesses, and to present evidence, as provided in the Act and the above referenced regulations.

ISSUES

The issues in this case are:

1. Whether miner worked at least 32 years in or around one or more coal mines;

2. Whether the miner has pneumoconiosis as defined by the Act;

3. Whether the miner's pneumoconiosis arose out of coal mine employment;

4. Whether the miner is totally disabled;

5. Whether the miner's disability is due to pneumoconiosis; and

6. Whether the evidence establishes a change in conditions and/or that a mistake was made in determination of any fact in the prior denial under § 725.310.

(DX 93). At the hearing, Employer withdrew as contested issues regarding whether miner's claim was timely filed, whether Claimant was a miner, whether Claimant has one dependent for purposes of augmentation, whether Employer is properly designated as responsible operator, and whether Claimant's most recent period of cumulative coal mine employment was with Employer. (DX 93; Tr. 7)

Based upon a thorough analysis of the entire record in this case, with due consideration accorded to the arguments of the parties, applicable statutory provisions, regulations, and relevant case law, I hereby make the following:

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background

Charles Byrge ("Claimant") was born on April 24, 1933; he was 72 years-old at the time of the hearing. (DX 1). On November 14, 1952, he married Colleen Carlson, and they remained married at the time of the hearing. (Tr. 27). I find that she is a dependent for purposes of augmentation. Claimant completed and signed interrogatories that were propounded by Employer on March 19, 2001. Claimant alleged 36 years of coal mine employment, beginning in

[2] In this Decision, "DX" refers to the Director's Exhibits, "EX" refers to the Employer's Exhibits, "CX" refers to the Claimant's Exhibits, and "Tr" refers to the official transcript of this proceeding.

1952 and lasting until 1993. He identified his last coal mine employment position as water tractor operator, which required him to sit for seven hours per day and to stand for one hour a day.

Claimant testified at the hearing that he was employed by Plateau Mining Corporation from September 1978 until he took a disability retirement on December 17, 1998. (Tr. 27). Claimant's last coal mine employment position, which lasted the last one-and-one-half years of his employment, was as a water truck driver in underground coal mining. (Tr. 28). As a water truck driver, Claimant was required to climb off and on the truck and walk 300 feet to reach the truck. (Tr. 37). Claimant stated that there were no aspects of his position as a water truck driver that were physically demanding, and he did not actually do any lifiting. (Tr. 37). Before he was a water truck driver, Claimant worked as a roof bolter for 13 years. (Tr. 28).

Claimant identified Dr. Potter as the physician who had been treating him since 1988. (Tr. 43). Claimant testified that he had been using supplemental oxygen since at least 1994. (Tr. 44). In 1984, Claimant stated that he underwent surgery for the placement of a stent for treatment of coronary artery blockage, followed subsequently by the placement of two more stents. (Tr. 44, 45).

Procedural History

Claimant filed an initial claim for benefits under the Act on February 14, 1994. (DX 1). The Director, Office of Workers' Compensation Programs ("OWCP") awarded benefits on November 2, 1994. (DX 29). Employer requested a formal hearing before the Office of the Administrative Law Judges. (DX 30). After conducting a formal hearing, Administrative Law Judge Roketenetz issued a decision and order – award of benefits on January 9, 1996. (DX 38). Administrative Law Judge Roketenetz found that the evidence supported the parties' stipulation that Claimant engaged in at least 15 years of coal mine employment. Employer did not contest its identity as the proper responsible operator and it was determined to be liable for benefits. Administrative Law Judge Roketenetz determined that the chest x-ray and biopsy evidence established the presence of pneumoconiosis under § 718.202(a), and then found that Claimant's pneumoconiosis arose out of coal mine employment under §718.203(b). Based on a the results of an arterial blood gas study from March 9, 1994 and the narrative medical opinion of Dr. Lincoln, Administrative Law Judge Roketenetz found that Claimant was totally disabled under the previous version of § 718.204(c). Based on Dr. Lincoln's opinion, Administrative Law Judge Roketenetz found that Claimant's totally disabling respiratory impairment was due to Claimant's pneumoconiosis arising out of coal mine employment. Accordingly, benefits were awarded to Claimant, augmented on behalf of his dependent wife, commencing on February 1, 1994.

Employer filed an appeal with the Benefits Review Board ("Board") on February 1, 1996. (DX 39). On October 18, 1996, Employer requested that the Board dismiss its petition for review pending the outcome of Employer's request for modification that it had filed with the OWCP that same day. (DX 40, 41). The Board granted Employer's request to dismiss its appeal on October 24, 1996, and remanded that claim to the OWCP for adjudication of Employer's modification request. (DX 42). On December 20, 1996, the OWCP issued a proposed decision

and order on remand denying Employer's request for modification. (DX 44). Employer requested a formal hearing before the Office of the Administrative Law Judges. (DX 45).

After conducting a formal hearing, Administrative Law Judge Mosser issued a decision and order denying benefits on June 29, 1999. (DX 64). Administrative Law Judge Mosser credited Claimant with 15 years of coal mine employment. Based on newly submitted chest x-rays and narrative reports concerning the biopsy report, Administrative Law Judge Mosser determined that a mistake in determination of fact occurred in the prior decision and order awarding benefits because the newly submitted evidence established that pneumoconiosis was not present. Since the totality of the medical evidence established the absence of pneumoconiosis, Administrative Law Judge Mosser granted Employer's request for modification of the prior finding of the presence of pneumoconiosis under § 718.202(a). Administrative Law Judge Mosser denied Employer's request to modify the prior finding that Claimant was totally disabled under the former version of § 718.204(c), determining that the totality of the evidence still supported a finding that Claimant was totally disabled. Administrative Law Judge Mosser also found that a mistake in determination of fact occurred regarding the causation of Claimant's total disability because the newly submitted evidence established that Claimant's total disability was due to other conditions. Since he found that benefits were mistakenly awarded, Administrative Law Judge Mosser determined that Claimant's claim for benefits under the Act must be denied.

On November 1, 1999, Claimant filed a request for modification. (DX 67). The OWCP issued a proposed decision and order denying Claimant's request for modification on January 12, 2000. (DX 67). Claimant did not appeal. Claimant again filed a request for modification on December 18, 2000. (DX 69, 70). On January 29, 2001, the OWCP issued a proposed decision and order denying Claimant's request for modification. (DX 70). Claimant did not appeal. On March 2, 2001, Claimant filed a request for modification. (DX 88). On September 23, 2002, the OWCP issued a proposed decision and order denying Claimant's request for modification. (DX 88). On September 30, 2002, Claimant requested a formal hearing before the Office of the Administrative Law Judges. (DX 89). The OWCP transferred this claim to the Office of the Administrative Law Judges on December 10, 2002 for a formal hearing. (DX 93).

Administrative Law Judge Roketenetz issued a notice of hearing, scheduling the formal hearing for July 17, 2003, in Price, Utah. (ALJX 1, 2). Employer filed a motion to compel discovery on June 9, 2003. Administrative Law Judge Roketenetz issued an order to show cause on June 11, 2003, ordering the parties to show cause within ten days why Employer's motion to compel should not be granted. On June 23, 2003, Administrative Law Judge Roketenetz issued an order reassigning the case to the undersigned. (ALJX 3). The formal hearing was conducted on July 17, 2003. Claimant offered five exhibits into the record, which the undersigned admitted. Employer offered 17 exhibits into the record. (Tr. 8, 9). The undersigned admitted Employer's exhibits 1-13, 15, and 17 into the record. In response to motions that the parties had submitted prior to and during the formal hearing, the undersigned issued an order on October 1, 2003 excluding the depositions of Drs. Renn and Farney (Employer's exhibits 14 and 16) and an order admitting the August 14, 2003 report of Dr. James as Claimant's exhibit 6. The undersigned's order granted Employer 30 days to submit an appropriate response to Dr. James' report as Employer's exhibit 18. On October 15, 2003, Employer filed a motion for

reconsideration of the undersigned's order excluding the depositions of Drs. Renn and Farney. Claimant objected to Employer's motion for reconsideration. On October 30, 2003, Employer filed Dr. Tuteur's third supplemental report to his prior examination report "in accordance with this Court's order." On October 31, 2003, Employer filed the October 28, 2003 report of Dr. Rosenberg in response to Dr. James' clarification report. Also on October 31, 2003, Claimant filed its closing brief. The undersigned issued an order granting Employer's motion for reconsideration on November 4, 2003. The depositions of Drs. Renn and Farney were admitted into the record and the parties were provided with an extension of time to file closing briefs.[3] Claimant submitted a letter on November 5, 2003 objecting to Employer's submission of the Dr. Rosenberg's October 28, 2003 report on the basis that it was contrary to the undersigned's October 1, 2003 order that allowed Employer to submit a response to Dr. James' clarification report. Claimant alleged that Dr. Rosenberg's October 28, 2003 report exceeded the scope of response that the undersigned permitted Employer to obtain. On November 18, 2003, Employer responded to Claimant's objection to the submission of Dr. Rosenberg's report, arguing that "[n]o unfair advantage was taken by the Employer in responding since Dr. James' earlier report was necessary for clarification of his earlier report." In the alternative, Employer requested that the undersigned keep Dr. Rosenberg's opinion associated with the file as an offer of proof under Fed.R.Evid. 103(a)(2) and 29 C.F.R. § 18.44(e). Employer filed a closing brief on November 24, 2003.

Dr. James' August 14, 2003 clarification report merely explained a discrepancy between findings listed on a B-reading form and his written report from June 25, 2003. Dr. James stated that he had reviewed his June 23, 2003 report and he determined that the clarifications he made regarding the February 26, 2002 chest x-ray did not significantly change the opinions outlined in his report. The October 27, 2003 report of Dr. Tuteur, entitled "Independent Medical Review (Third Supplement)", reviewed Dr. James' reports of June 25, 2003 and August 14, 2003. Dr. Tuteur noted that Dr. James' August 14, 2003 report clarifies his readings of standard chest radiographs. Dr. Tuteur then criticized several of the conclusions drawn by Dr. James in his June 25, 2003 report. Beyond noting the clarification made by Dr. James, Dr. Tuteur did not specifically address the clarification made by Dr. James. Since Dr. Tuteur's October 27, 2003 report did not address the effect, if any, that Dr. James' clarification report had on his own opinion, I find that Dr. Tuteur's October 27, 2003 report is not an appropriate response to Dr. James' clarification report. Therefore, I exclude Dr. Tuteur's October 27, 2003 report from the record.

Employer also submitted the October 28, 2003 report of Dr. Rosenberg in response to Dr. James' August 14, 2003 clarification report.[4] Dr. Rosenberg's discussion detailed his criticisms of Dr. James' June 25, 2003 report. He only referenced Dr. James' August 14, 2003 clarification report to the extent that he noted that the clarifications did not alter Dr. James' overall opinion. I

[3] Claimant's objections to Drs. Renn's and Repsher's use of scientific medical articles, as contained in the deposition testimony of Dr. Repsher, are rendered moot by the undersigned's order admitting the deposition testimony of Drs. Renn and Farney.

[4] The undersigned's October 1, 2003 order granted Employer 30 days to submit an appropriate response to the August 14, 2003 clarification report of Dr. James. It did not grant Employer 30 days to submit appropriate response*s* to the August 14, 2003 clarification report of Dr. James. Neither of the purported responses addressed the effect, if any, that Dr. James' clarification had on their prior opinions. Employer misused the undersigned's order as an opportunity to develop and submit medical reports that criticized Dr. James' June 25, 2003 report.

admit Dr. Rosenberg's October 28, 2003 report to the limited extent that he opines that Dr. James' clarification report did not alter his original June 25, 2003 report. I exclude the discussion section of Dr. Rosenberg's October 28, 2003 report because it does not address the clarification report.

Length of Coal Mine Employment

The parties previously stipulated that Claimant engaged in at least 15 years of coal mine employment. Administrative Law Judge Roketenetz determined that the record supported the parties' stipulation. Administrative Law Judge Mosser also found that Claimant engaged in 15 years of coal mine employment. In this modification proceeding, Employer identified the issue of whether Claimant engaged in 32 years of coal mine employment as a contested issue. At the hearing, Claimant testified that he began coal mine employment in June of 1952. Neither party addressed provided any argument in their closing brief as to why they should not be bound by their previous stipulation. However, I find that there is substantial evidence in the record to support the parties' stipulation that Claimant engaged in 15 years of coal mine employment.

On the initial Employment History form that Claimant completed, he listed the beginning of his coal mine employment as June 1952. (DX 2). He continued to engage in coal mine employment through October of 1959. Claimant returned to coal mine employment in January 1966 until he stopped again in April 1972. Claimant began coal mine employment again in April 1974, and he continued to engage in coal mine employment until December 1977. Claimant then worked for Employer from September 1978 until December 1993. However, Claimant's Social Security Earnings records reflect substantial gainful employment for non-coal mine related employers from 1952 through 1976. The record fails to substantiate Claimant's account of 32 years of coal mine employment, but it does substantiate Claimant's employment with Employer from 1978 through 1993. Therefore, I find that Claimant engaged in at least 15 years of coal mine employment.

Responsible Operator

Liability under the Act is assessed against the most recent operator which meets the requirements of §§ 725.494 and 725.495. Employer does not and has not contested its identity as the employer with whom Claimant spent his last cumulative one year period of coal mine employment. Prior to the hearing, Employer filed a letter dated May 3, 2003, specifically withdrawing its controversion to being identified as the proper responsible operator. (DX 96). Therefore, I find that Plateau Mining Corporation is properly designated as the responsible operator in this case.

MEDICAL EVIDENCE

Section 718.101(b) requires any clinical test or examination to be in substantial compliance with the applicable standard in order to constitute evidence of the fact for which it is proffered. *See* §§ 718.102 - 718.107. The prior denial of Claimant's claim most recently occurred on June 29, 1999. Therefore, the undersigned will consider any evidence developed after June 29, 1999 for the purposes of determining whether a change in conditions has occurred.

I incorporate by reference, as if fully rewritten herein, the chest x-rays, pulmonary function tests, arterial blood gas studies, narrative medical reports, and biopsy reports contained in the decisions and orders of Administrative Law Judges Roketenetz and Mosser, to the extent that they are not inconsistent with evidence summarized herein.

On February 21, 2003, Employer submitted a letter with Employer's Exhibits 2-7 attached. Employer's Exhibits 2-7 were submitted as exhibits that Employer determined should have been included in the Director's exhibits. The undersigned finds that the records submitted by Employer as Employer's Exhibits 2, 3, 5, 6, and 7 are properly admissible. However, the undersigned excludes Employer's Exhibit 4 from the record. Employer describes Employer's Exhibit 4 as a "95 page exhibit from IHC Hospitals of Utah County (originally tendered as Employer's Exhibit 19)." The first page of Employer's Exhibit 4 is a letter dated November 16, 1996, addressed to the former counsel for Employer, and written by a senior claims examiner for the OWCP. The claims examiner returned the medical records that presently comprise Employer's Exhibit 4 to the former counsel for Employer because he found the records to be irrelevant and highly prejudicial to the Claimant. Since the Director, OWCP returned the records contained in Employer's Exhibit 4 to the Employer, they should not have been included in the Director's Exhibits. Upon review of Employer's Exhibit 4, the undersigned finds that the Director, OWCP properly excluded these medical records from the record and properly returned them to Employer. Employer's Exhibit 4 contains records of psychiatric treatment provided to Claimant by the Utah Valley Regional Medical Center in January 1989. The only relevance the psychiatric records possibly have to this proceeding is that they contain Claimant's accounts of his coal mine employment and smoking history, as well as Claimant's self-reporting that he has Black Lung. The highly prejudicial nature of the psychiatric treatment records outweighs any potentially limited degree of relevant information contained in the records. The nature of the records and the fact that these records are from 1989 engenders the records with little, if any, probative weight towards determining whether a mistake in fact occurred or whether a change in conditions has occurred since the June 29, 1999 decision and order. In light of the potential for harm, not only to Claimant but also to the persons identified in the records who are not parties to this claim, the undersigned excludes Employer's Exhibit 4.

X-RAYS

Exhibit	**Date of X-ray**	**Date of Reading**	**Physician / Credentials**	**Interpretation**
CX 3	12/23/97	6/12/03	James, B-reader	1/0
EX 10	12/23/97	6/05/03	Renn, B-reader	negative
CX 3	5/15/00	6/12/03	James, B-reader	1/0
DX 80	5/04/01	5/05/01	James, B-reader	1/0

DX 81	5/04/01	8/04/02	Preger, BCR[5], B-reader[6]	Film quality 1
DX 87	5/4/01	8/26/02	Wiot, BCR, B-reader	negative
DX 91	5/4/01	10/29/02	Spitz, BCR, B-reader	negative
EX 1	5/4/01	11/4/02	Shipley, BCR, B-reader	negative
DX 81	5/4/01	12/8/02	Preger, BCR, B-reader	negative
DX 76	2/26/02	3/1/02	Mann,	negative
DX 76	2/26/02	5/29/02	Wiot, BCR, B-reader	negative
CX 3	2/26/02	6/12/03	James, B-reader	0/1
DX 82	2/26/02	6/26/02	Shipley, BCR, B-reader	negative
DX 86	2/26/02	8/3/02	Spitz, BCR, B-reader	negative
EX 10	2/26/02	6/5/03	Renn, B-reader	negative

The chest x-rays obtained on September 21, 1998 and January 21, 2000 were not interpreted in accordance with the requirements of § 718.102 or Appendix A to Part 718. (EX 5). The undersigned cannot infer whether or not the films are positive or negative for the existence of pneumoconiosis.

PULMONARY FUNCTION TESTS

Exhibit/ Date	**Co-op./ Undst./ Tracings**	**Age/ Height**	**FEV_1**	**FVC**	**MVV**	**FEV_1/ FVC**	**Qualifying Results**
DX 80 5/4/01	Acceptable/ Reproducible/ Yes	68 70”	2.18	2.79		78%	No
DX 76 2/26/02	Good/ Good/ Yes	68 70”	2.45 2.76*	3.56 3.69*		69% 75%*	No No

* post-bronchodilator values

ARTERIAL BLOOD GAS STUDIES

Exhibit	**Date**	**pCO_2**	**pO_2**	**Qualifying**
DX 80[7]	5/4/01	34.4	51.5	Yes

[5] A physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. *See* 20 C.F.R. § 727.206(b)(2)(III). The qualifications of physicians are a matter of public record at the National Institute of Occupational Safety and Health reviewing facility at Morgantown, West Virginia.

[6] A “B” reader is a physician who has demonstrated proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by or on behalf of the Department of Health and Human Services. This is a matter of public record at HHS National Institute for Occupational Safety and Health reviewing facility at Morgantown, West Virginia. (42 C.F.R. § 37.5l) Consequently, greater weight is given to a diagnosis by a "B" Reader. *See Blackburn v. Director, OWCP*, 2 B.L.R. 1-153 (1979).

Alt. over 5, 999		32.1*	46.7*	Yes
DX 76 Alt. 4450 feet	2/26/02	34.8	55.9	Yes

* results obtained after exercise

Narrative Reports

Sterling Potter, M.D., who is board-certified in family practice, issued a narrative report on October 25, 1999. (DX 66). Dr. Potter stated that Claimant had pulmonary disease related to his coal mine employment. He noted that Claimant was using supplemental oxygen every day when he lied down. Dr. Potter opined that Claimant's lung disease is related to and worsened by his coal mine experience of over 30 years of working at the face. Dr. Potter then stated that Claimant was being evaluated for CWP disability. In the strictest sense of the law, Dr. Potter found that Claimant "qualifies for this in as much as his coal mine experience has contributed to his lung disease."

Dr. Potter issued another letter on December 4, 2000. (DX 69). He noted that Claimant continued to have chronic obstructive lung disease ("COPD"). Dr. Potter found that Claimant's lung disease was made worse by his 30 year history of coal mine employment that was mostly at the face. Dr. Potter added that Claimant had not smoked cigarettes for 22 years. He then stated that Claimant's exposure to a coal mine environment was a significant factor to his lung disease. Dr. Potter opined that Claimant's coal dust exposure has contributed to his chronic lung disease and continued need for supplemental oxygen when lying down.

David James, M.D. examined Claimant on May 4, 2001. (DX 80). He noted that Claimant retired in 1992 after engaging in 37 years of coal mine employment, last working as a water truck driver. Dr. James identified the exertional requirements of Claimant's work roof bolting job, which he held for 13 years before driving a water truck, as requiring lifting of 40 to 50 pounds and walking up to two miles per day. Dr. James found Claimant's past medical history to be notable for diabetes with peripheral neuropathy, elevated cholesterol, history of carotid artery disease, history of coronary artery disease with angioplasty on multiple occasions, and a pulmonary embolism in 1995 that required anticoagulation for several months. He documented a smoking history beginning at the age of 18 and lasting until 1978 in the amount of one-and-one-half packs per day, which amounted to a 25 pack-year history when periods where Claimant was not smoking were subtracted. He also noted that Claimant's wife reported that Claimant had several months of sleep apnea spells. Upon physical examination, Claimant's lungs were clear to auscultation, but they revealed slightly decreased breath sounds. Dr. James observed that Claimant was heavy-set and experienced dyspnea after walking to the exam table. He submitted Claimant to a chest x-ray, PFT, ABG, and an exercise study. He opined that the chest x-ray revealed parenchymal abnormalities consistent with pneumoconiosis. Dr. James noted that Claimant had difficulty performing the lung volume part of the PFT, rendering them

[7] Timothy Kennedy, M.D., who is board-certified in internal medicine and the subspecialties of pulmonary disease and critical care, completed a Department of Labor Validation of Pulmonary Function and Arterial Blood Gas Study form on July 29, 2002 regarding the May 4, 2001 ABG. (DX 82). He found the resting and exercise studies to be technically acceptable.

uninterpretable. He interpreted the ABG and exercise test as revealing low exercise tolerance, low PO_2 at rest with mild decrease after exercise, and no significant ECG changes with exercise. Dr. James impression was that Claimant suffered from a diffuse lung disease that was most prominent in the lower lung zones. He referenced the abnormal CT scan from 1997 and a transbronchial biopsy showing fibrosis. Dr. James commented that the changes in Claimant's lungs were consistent with diffuse fibrotic disease. He commented that Claimant had no known exposure to asbestos. He opined that Claimant suffered from diffuse pulmonary fibrosis secondary to chronic inhalation of coal mine dust, adding that CWP can present with more prominent lower lobe interstitial disease as opposed to the more common upper lobe, rounded opacities form of CWP. Dr. James also found that Claimant might be suffering from sleep apnea based on his wife's observations. Dr. James concluded that Claimant would not be able to perform the usual jobs that he conducted as a coal miner, leaving him totally disabled as a coal miner. Claimant's diffuse fibrotic disease, based in part on Claimant's low vital capacity, low diffusing capacity, and exercise induced hypoxemia, would be severe enough to prevent Claimant from performing his usual coal mine employment. He found that Claimant should use supplemental oxygen 24 hours per day due to the significant oxygen desaturation with minimal activity.

Robert Farney, M.D. examined Claimant on February 26, 2002. (DX 76). He previously examined Claimant on December 23, 1997. Dr. Farney conducted a physical examination, and he reviewed the results of an arterial blood gas study, pulmonary function test, and a chest x-ray and CT scan interpreted by Dr. Maan. Claimant reported shortness of breath on exertion. Since Dr. Farney last examined Claimant, Claimant developed several medical problems, including a coronary angioplasty and stent placement in May 2001 for coronary artery disease, left internal carotid artery stenosis diagnosis in January 1999 by Dr. Clayson, and expressive aphagia and dysarthia that had resolved leaving only a residual problem of memory impairment. Dr. Farney noted a past history of gastroesophageal reflux, numbness in his legs related to disk disease, and poor quality sleep. On physical examination, Dr. Farney found Claimant to be obese with no overt respiratory difficulties. Upon examination of Claimant's chest, Dr. Farney detected mild basilar crackles, primarily in the left lung base. He found the ABG he conducted to be normal for Claimant's age and the elevation. Dr. Farney determined that the prebronchodilator PFT was consistent with mild airflow obstruction followed by a positive bronchodilator response. He did not find any evidence of a restrictive disease from the PFT. Claimant's diffusing capacity showed marked reduction consistent with interstitial lung disease or emphysema. Dr. Farney reviewed Dr. Maan's interpretation of a chest x-ray and CT scan, noting that they were negative for CWP or fibrosis. An EKG revealed sinus bradycardia and ST-T wave abnormalities. Dr. Farney concluded that Claimant did not have CWP or any other pulmonary disease due to coal dust exposure. He found that Claimant had a significant reduction of the diffusion capacity, which is most likely secondary to emphysema associated with tobacco smoke. Dr. Farney also noted that Claimant has co-morbid conditions of coronary artery disease, insulin dependent diabetes, obesity and probably obstructive sleep apnea. He found that Claimant's exertional limitations are due to these multiple conditions, none of which are related to coal dust exposure.

David Rosenberg, M.D., who is board-certified in internal medicine and occupational medicine and the subspecialty of pulmonary disease, issued a consultative report on April 28, 2003. (EX 8). Dr. Rosenberg reviewed and summarized Claimant's employment records,

medical records from 1994, 1997, 2001, and 2002, hospital records, and narrative medial reports from 1992 through 2002. He considered that Claimant engaged in 32 years of coal mine employment as a face boss, miner operator, rope rider, and shuttle car operator. Claimant last engaged in coal mine employment in December 1993. In addition to Claimant's extensive smoking history, Dr. Rosenberg noted that Claimant's medical history involved diabetes with peripheral vascular disease, coronary artery disease, hiatal hernia with reflux, depression, and alcoholism. Claimant began experiencing shortness of breath in 1993. Dr. Rosenberg noted that Claimant's lungs have generally been clear on physical examination and his chest x-rays have been variably interpreted as either being negative or having some basilar linear opacities. He added that the CT scans have not demonstrated any micronodularity, nor did the biopsy. Dr. Rosenberg noted that the chest x-ray findings of basilar linear opacities are inconsistent with the upper lobe micronodular changes associated with the past inhalation of coal dust. He also commented that chronic end-respiratory rales have not been detected on auscultation of Claimant's chest, and Claimant's lung volume measurements have been normal without restriction. However, Dr. Rosenberg found that the increasing A-a gradient with exercise indicates some interstitial involvement. Based on the character of the linear opacities with basilar involvement, Dr. Rosenberg concluded that it is not the pattern of interstitial lung disease associated with coal dust exposure. He opined that this type of development can develop consequent to cigarette smoking. Dr. Rosenberg expressly found that Claimant did not suffer from the interstitial form of CWP.

Dr. Rosenberg found that Claimant did not suffer from any pulmonary restriction, but he did find that Claimant had an increasing gas exchange abnormality with exercise. Additionally, he noted that Claimant appears to have a right to left shunt, which is an oxygenation abnormality that would render Claimant unable to perform his usual coal mine employment. Dr. Rosenberg opined that shunt formation does not develop in the setting of interstitial lung disease. Interstitial lung disease results in ventilation perfusion mismatch or diffusion abnormalities, which correct consequent to the administration of 100% oxygen. He opined that Claimant's shunt formation, which he found to probably be the major contributing factor to Claimant's disability, is not the consequence of past coal dust exposure and the presence of interstitial lung disease. Dr. Rosenberg stated that Claimant probably has an arterial-venous malformation someplace in his body, potentially in his liver, that is causing the shunt. He added that the combination of Claimant's coronary artery disease and peripheral vascular disease, which are unrelated to coal mine dust exposure, render Claimant disabled. Dr. Rosenberg agreed that coal mine dust exposure can cause COPD that begins in a focal fashion around the coal macule, but he noted that Claimant's evaluations have not revealed any evidence of micronodularity. Consequently, Dr. Rosenberg opined that any minimal obstruction that Claimant suffers from is not related to coal mine dust exposure. Instead, he attributed Claimant's minimal degree of obstruction to Claimant's cigarette smoking history. Dr. Rosenberg concluded that Claimant does not suffer from CWP or associated impairment. While he found Claimant to be totally disabled, he opined that it was not caused by the inhalation of coal mine dust. Rather, his disability is due to a shunt formation and some nonspecific interstitial lung disease quite likely related to his past smoking history.

Joseph Renn, III, M.D., who is board-certified in internal medicine and the subspecialty of pulmonary disease, issued a consultative report on June 9, 2003. (EX 10). Dr. Renn

delineated the 33 categories of medical evidence that he reviewed and summarized in his report. He stated that Claimant was a 70 year-old who has chronic hypoxemia owing to recurrent pulmonary emboli. Dr. Renn noted that Claimant began coal mine employment in 1952 and ended in 1993 after performing work as a shuttlecar operator, roof bolter, and lastly as a water wagon driver. From the medical records, he considered a smoking history that ranged from 15 to 87 pack-years. Dr. Renn provided an accurate summary of Claimant's past medical history. He noted that physical examinations occasionally detected diminished breath sounds and bibasilar crackles, adding that most examinations of the chest were normal. Dr. Renn found that Claimant's weight was severely overweight. From his review of PFT studies from 1989 through 2001, he opined that the ventilatory function represented by the somewhat valid studies is normal. Dr. Renn added that the four lung volume studies reveal a normal total lung capacity, which eliminates the presence of a restrictive ventilatory defect. He found that the diffusing capacity study from February 26, 2002 was invalid based on the graph tracings, that the May 4, 2001 and December 23, 1997 studies were mildly reduced but correct completely to normal when considering alveolar volume, and that the December 23, 1993 and August 1, 1992 studies were normal.

Dr. Renn noted that Claimant has had multiple ABGs performed since 1984. By 1987, Claimant had developed mild resting hypoxemia that has generally persisted. He added that the two exercise-ABGs revealed exercise-induced hypoxemia. Dr. Renn commented that one ABG, conducted while Claimant was on 100% oxygen revealed an alveolar-arterial oxygen gradient indicative of a fairly marked shunt. He opined that Claimant's carboxyhemoglobin level from the October 21, 1987 and May 4, 2001 ABGs was 2.1%, which is consistent with a person who is smoking one-half packs of cigarettes per day in comparison to the 1.5% range for non-smokers. Dr. Renn reported that he reviewed the CT scans of December 23, 1997 and February 26, 2002, finding them to contain no changes consistent with CWP. He did find nonspecific minimal bibasilar interstitial fibrotic changes. He interpreted a ventilation-perfusion lung scan conducted on December 22, 1993 to be consistent with a finding of COPD. Dr. Renn also commented that the February 5, 1995 pulmonary angiography revealed pulmonary emboli at two separate sites in the right lung. Dr. Renn's impression was that Claimant had chronic hypoxemia owing to recurrent pulmonary emboli.

Under the heading of respiratory system, Dr. Renn diagnosed: (1) chronic hypoxemia owing to (2) recurrent pulmonary emboli; (3) status post right lower lobe pulmonary infarction; (4) a pneumoconiosis does not exist; (5) mild decrease of diffusing capacity owing to #2 above; (6) normal dynamic and static ventilatory function. Under the heading of cardiovascular system, Dr. Renn diagnose: (1) atherosclerotic coronary vascular disease manifested by (2) angina pectoris and the necessity for (3) multiple PTCAs and stent placements; (4) systemic hypertension; and (5) atherosclerotic peripheral vascular disease. Dr. Renn made the following diagnoses under the heading of metabolic/endocrine system: (1) adult onset diabetes mellitus; (2) severe exogenous obesity; and (3) hypercholesterolemia. Additionally, Dr. Renn diagnosed secondary polycythemia owing to #1 under respiratory system, GERD owing to esophageal hiatus hernia, and diabetic peripheral neuropathy. Dr. Renn concluded that none of the above listed diagnoses were caused or contributed to by Claimant's exposure to coal mine dust. He found that Claimant's chronic hypoxemia owing to recurrent pulmonary emboli was not caused or contributed to by Claimant's exposure to coal mine dust. He found that Claimant is totally

disabled and unable to perform his usual coal mining job or any job requiring similar effort due to his chronic hypoxemia owing to recurrent pulmonary emboli.

Peter Tuteur, M.D., who is board-certified in internal medicine and the subspecialty of pulmonary disease, issued a consultative opinion on June 16, 2003. (EX 12). Dr. Tuteur stated that his consultative report was based on his independent medical review from May 6, 1998, his supplemental May 16, 1998 report, and a delineated list of Claimant's subsequent medical records. He noted that Claimant worked in the coal mining industry for three decades, predominantly in underground mining at the face. Dr. Tuteur noted a cigarette smoking history that was documented as beginning in Claimant's teens and lasted until beyond the age of 50 in an amount as much as one-and-one-half packs per day. He listed a past medical history notable for arteriosclerotic heart disease, diabetes mellitus, overweight status, and an episode of major depression, as well as peptic ulcer disease and carpal tunnel syndrome. Dr. Tuteur found Claimant's most significant prior clinical problem to be that of progressive and severe breathlessness not regularly associated with cough, expectoration, wheezing, or chest pain. He found that physical examination of Claimant's chest was most often normal. Despite normal spirometry and lung volumes, Claimant had a mild resting impairment of gas exchange with normal ventilation that became dramatically worse during exercise and was not corrected by administration of 100% oxygen. Dr. Tuteur commented that chest x-rays were often interpreted as free of changes compatible with CWP. He added that a CT scan of the thorax was interpreted as demonstrating subtle bilateral lower lung field interstitial pulmonary fibrosis with some adenopathy. Dr. Tuteur noted that a transbronchial biopsy only demonstrated the presence of some pigmented dust, but no tissue reaction to foreign material.

Dr. Tuteur commented that the newly considered medical evidence was important in several aspects. First, he found that the detection of multiple pulmonary emboli in 1995 through a pulmonary angiography was important because it prevented an additional clinical condition from being potentially responsible for right to left shunting of blood producing the physiologic impairment demonstrated by extensive and longitudinal studies. Dr. Tuteur also commented that the extensive progressive coronary artery disease that required recurrent invasive supplementing medical management was the documented clinical course through mid 2001. He added that an additional manifestation of Claimant's peripheral vascular disease included a stroke that Claimant suffered in 1999 requiring left carotid angioplasty to relieve the stenosis. Dr. Tuteur found that Claimant's breathlessness and exercise tolerance became more severe, and that spirometry and lung volumes remained relatively stable. Claimant's shunt physiology was reaffirmed through May 2001 ABG that showed a decrease in pO2 with 100% breathing. Dr. Tuteur commented that additional chest radiographic evidence continues to be absent of changes compatible with CWP.

Based on the totality of all of the medical evidence, Dr. Tuteur opined that Claimant did not have CWP or any other coal mine dust-induced disease process that is of sufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary or respiratory function, or radiographic abnormalities. However, he found that Claimant was totally disabled to such an extent that he is unable to complete the responsibilities of a coal miner or work requiring similar effort. He added that Claimant does have impairment of respiratory function and clear cut mild gas exchange at rest that dramatically worsens during

exercise. He opined that Claimant's gas exchange limitations may be due to three conditions. First, areas of the lung with low ventilation to perfusion ratios allow some poorly oxygenated blood to skip through the capillary network and cause arterial hypoxemia in disease processes like COPD, but Dr. Tuteur excludes that as the cause in Claimant's situation because the DA-aO2 gradient did not narrow as the pO2 increased. He also ruled out the second possible cause of a pure diffusion problem due to an interstitial fibrotic process that operates and obliterates the capillary bed because there was no improvement in oxygen exchange with supplemental oxygen. Instead, Dr. Tuteur attributed the problem to Claimant's right to left shunt, which he defined as blood returning to the right side of the heart that flows in such a manner that it totally bypasses the gas exchange portions of the lungs. He stated that a right to left shunt can occur through an atrial or ventricular septal defect, the presence of an arterial venous malfunction in disease processes such as hereditary telangiectasia or vascular abnormalities associated with chronic cirrhotic liver disease, or through pulmonary emboli. He found it notable that Claimant drank alcohol heavily for 20-25 years of his life and had manifestations of hereditary telangiectasias in the form of nosebleeds (hospital records document epistaxis in 1998 and 2000), as well as a stroke and a rigorously documented pulmonary embolism. Therefore, Dr. Tuteur concluded that Claimant's physiologic impairment contributing to severe exercise limitation is not one seen with the pathologic process of CWP or the obstructive abnormality associated with the inhalation of coal dust. Accordingly, Dr. Tuteur opined that Claimant did not have CWP or any other coal mine dust-induced disease process of sufficient severity and profusion to produce clinical symptoms, physical exam abnormalities, physiologic impairment, or radiographic change. Dr. Tuteur then opined that Claimant's clinical symptomatology, which he determined to be of sufficient severity and magnitude to render Claimant disabled from returning to work in the coal mine industry or work requiring similar effort, is a result of not only the coronary artery disease that required extensive therapeutic intervention, but also due to the physiologic process of worsening gas exchange during exercise due to the presence of a right to left shunt. He could not identify the specific anatomical site of the shunt. Dr. Tuteur added that Claimant's significant pulmonary and respiratory impairment was not caused, related to, or contributed to by the inhalation of coal mine dust or the development of CWP.

On June 16, 2003, Dr. Potter authored a letter to counsel for Claimant. (CX 1). Dr. Potter stated that he had been treating Claimant since 1988. Since the beginning of his treatment, Claimant has developed progressive lung disease requiring oxygen. Dr. Potter stated that the major factor in Claimant's decision to stop working was his lungs. He referred Claimant to Dr. Lincoln, a pulmonary specialist. Dr. Potter noted that Claimant had a smoking history of 30-40 packs years. He opined that Claimant's smoking and the dust in his principal job as a roof bolter contributed to Claimant's pulmonary disability. He commented that a CT scan showed pulmonary fibrosis. Dr. Potter noted that, despite Claimant's relatively normal pulmonary function tests, Claimant demonstrated dyspnea on exertion and hypoxia. Claimant's pulse oximetry and arterial blood gas studies clearly demonstrate that Claimant is disabled due to his lungs. Claimant's hypoxia alone is sufficient to prevent Claimant from working on a mine rescue team or even to work in a coal mine with his current pulmonary status. Dr. Potter noted that Claimant had multiple other medical problems that preclude him from engaging in heavy labor of any kind, including coronary artery disease, diabetes, peripheral vascular disease. Nevertheless, Dr. Potter found that Claimant's lungs were the major contributing factor leading Claimant to stop working in the mines. On numerous x-rays, Dr. Potter noted that Claimant

demonstrated pulmonary fibrosis which is significantly related to or substantially aggravated by dust exposure as well as Claimant's cigarette smoking history. Dr. Potter found it difficult to believe that working for 13 years as a roof bolter could not substantially contribute to fibrosis, again noting that Claimant's smoking history is also a contributory factor. Dr. Potter opined that it appeared that Claimant's pulmonary function has deteriorated over the past few years based on his comparison of a 1994 PFT and a 2002 PFT. He found a significant change in the COPD experienced by Claimant based on all of the PFTs conducted by the Salt Lake Clinic. Dr. Potter added that Claimant's pulmonary deterioration had occurred despite Claimant not smoking during that time period. Dr. Potter concluded, based on his 14 years of treatment, his review of the chest x-rays, PFTs, and ABGs, that Claimant suffered from simple CWP with fibrosis descending into the lower lobes. He also concluded that Claimant's pulmonary disease led to his retirement, adding that Claimant is, without question, currently disabled from working in a coal mine. Lastly, Dr. Potter concluded that Claimant's pulmonary function has deteriorated in the last few years as demonstrated by Claimant's increasing obstructive lung disease as noted in 2001 and 2002.

David James, M.D., who is board-certified in internal medicine and the subspecialties of pulmonary disease and critical care, issued a consultative report on June 25, 2003. (CX 3). Dr. James examined Claimant on May 4, 2001, and he reviewed Claimant's medical records that included Dr. Rosenberg's April 28, 2003 report, Dr. Renn's June 5, 2003 report, and additional chest x-rays and CT scans. Dr. James reviewed chest x-ray interpretations from films obtained in 1998 through 2002. He also rendered interpretations of chest x-rays dated December 23, 1997, May 15, 2000, and February 26, 2002. He found that the February 26, 2002 film was positive for the existence of pneumoconiosis. The copies of the CT scans from 1997 and 2002 that Dr. James reviewed were not of sufficient quality to allow for an interpretation. Dr. James then provided a response to five questions.

The first question Dr. James responded to was, "[i]s the radiographic and biopsy evidence consistent with medical or legal definition of pneumoconiosis or both?" Dr. James opined that Claimant has clinical evidence of pulmonary fibrosis based on the "radiographic, pulmonary function abnormalities, and gas exchange abnormalities." He noted that chest x-ray B-readings performed by himself and other B-readers contained in the record have observed abnormalities consistent with pneumoconiosis, even though not all B-readers found the presence of pneumoconiosis. Dr. James then referenced CT scan findings dating back to 1994 that noted minimal interstitial densities in Claimant's lower lung bases. He noted that the transbronchial biopsy from 1994 did not show evidence of CWP or any other fibrotic lung process. Dr. James did note that the presence of fibrosis was reported in the biopsy sample, but the specimen size limited the conclusions that could be drawn from the presence of the fibrosis. Over the past ten years, Dr. James determined that Claimant had suffered a worsening in his diffusing capacity to carbon monoxide based on normal studies from 1992 and 1993 followed by decreases in the DL_{CO} in 1997, 2001, and 2002. He commented that the most recent result was 46% of normal. Dr. James also found that Claimant had abnormal oxygenation with exercise and on some resting studies. The resting value from the ABG performed on May 14, 2001 was abnormally low with an abnormal decline in oxygenation with exercise. Dr. James concluded that the pulmonary

function and oxygenation abnormalities are more likely than not secondary to the diffuse lung disease that is observed on the radiographic studies and are due to the chronic inhalation of coal mine dust.

Secondly, Dr. James responded to the question of, "[d]oes Mr. Byrge have evidence of COPD and emphysema and does this fit the definition of legal pneumoconiosis?" He concluded that there is no consistent clinical evidence that Claimant has COPD or emphysema. From the February 26, 2002 spirometry, there was evidence of airflow obstruction. Dr. James noted that this was the only time airflow obstruction was documented between 1984 and 2002. The CT scans from 1994, 1997, and 2002 did not show evidence of emphysema. Instead, Dr. James attributed the significant decline in Claimant's diffusing capacity and oxygenation to his diffuse fibrotic lung disease. He added that he was aware that the record contained documentation of a smoking history up to a 45 pack-year history, which was greater than the history he obtained in 2001. However, he opined that such a difference does not significantly change his respiratory diagnosis.

The third question that Dr. James answered was, "[w]hat is the role of Mr. Byrge's coal mine dust in the development of his medical or legal pneumoconiosis, or both?" He opined that it was more likely than not that Claimant developed diffuse fibrotic lung disease from his long-term exposure to coal mine dust, which is a form of pneumoconiosis. Dr. James described the more common type of nodular CWP that develops in the upper lung zones of coal miners. He then described a fibrotic process, which develops first in the lower zones, that a minority of hard rock coal miners acquire. Dr. James stated that it is not well-understood why this form of pneumoconiosis develops instead of the more common nodular form, but he added that smoking may be contributing factor for the development of diffuse type of fibrotic disease resulting from exposure to inorganic dust. Dr. James referenced seven scientific medical studies. He then opined that it is unlikely that the radiographic and physiologic abnormalities observed in Claimant are due to another, non-coal mine dust induced abnormality. He noted that it is less likely that Claimant has COPD, but cautioned that only additional testing will identify whether the airflow obstruction detected in the February 2002 PFT is persistent and not due to an acute process such as bronchitis. Dr. James stated that the radiographic abnormalities he identified were not consistent with a diagnosis of COPD. Moreover, Dr. James opined that Claimant did not suffer from idiopathic fibrosis (fibrosis of unknown origin) because Claimant did not demonstrate the usual physical findings of someone with idiopathic pulmonary fibrosis (crackles on chest auscultation and clubbing of digits) and because he identified the suspected cause of Claimant's fibrosis as arising out of coal mine and hard rock dust. Dr. James ruled out sarcoidosis as a possible diagnosis because there was no pathological evidence of it in the 1994 biopsy, which is a very sensitive for detecting sarcoidosis. He also ruled out asbestosis because Claimant had no known history of exposure and there were no pleural changes consistent with asbestosis on the chest x-rays and CT scans. Dr. James found it unlikely that Claimant's coronary artery disease caused the pulmonary fibrosis because it doesn't generally result in long-term abnormal chest x-ray or gas exchange findings unless the coronary artery disease results in congestive heart failure. However, Dr. James noted that Claimant underwent several echocardiograms and cardiac catheterization, none of which revealed congestive heart disease. Dr. James acknowledged that Claimant was obese and that obesity can alter lung function in the form of decreased vital capacity. However, Claimant's vital capacity measurements have been

mostly normal. Dr. James also stated that obesity does not alter diffusing capacity. Dr. James noted that previous evaluations of Claimant included a procedure called a shunt study. He described a shunt as a one cause of low arterial oxygen level that occurs when deoxygenated blood mixes with oxygenated blood. Dr. James stated that a small shunt of 5% occurs in all healthy people. A shunt study from April 1, 1994 showed a 17% shunt and an October 20, 1994 study that identified a 6% shunt. Dr. James opined that Claimant has no evidence of the several causes of a shunt, which include congenital heart disease, lung cancer, pulmonary arteriovenous shunts, or shunts resulting from liver disease. There was also no evidence of pulmonary intravenous shunts on the CT scans.

The next question Dr. James addressed was, "[p]lease state whether the medical evidence supports your opinion that the Claimant had a total respiratory disability and whether Claimant's legal and medical pneumoconiosis, or both, is a contributing factor to his total respiratory disability." Dr. James answered that the respiratory impairment resulting from Claimant's diffuse pulmonary fibrosis would be rated as severe based on the abnormal diffusing capacity and the decline in arterial oxygen with exercise. He attributed Claimant's poor exercise tolerance that was demonstrated by cardiopulmonary exercise tests in 1997 and 2001, in part, to Claimant's diffuse pulmonary fibrosis. Dr. James found that Claimant's coronary artery disease, obesity, and possible deconditioning were factors contributing to his poor exercise tolerance. Based on the physical requirements, as he outlined in his May 14, 2001 report, and Claimant's diffuse fibrotic lung disease, Dr. James concluded that Claimant would not be able to perform his usual coal mine employment.

The fifth question Dr. James addressed was, "[p]lease state whether the Claimant's legal or medical pneumoconiosis [sic] dust related impairment has worsened since June 29, 1999." Dr. James responded by stating that there is pulmonary function test evidence of a worsening in Claimant's respiratory disease since 1999. He noted that the December 23, 1997 diffusing capacity value was 69% of normal. Studies from May 4, 2001 showed a diffusing capacity of 63%, and by February 26, 2002, it had dropped to 46% of normal.

Dr. Rosenberg was deposed on July 8, 2003. (EX 15). He reiterated the findings and conclusions contained in his April 28, 2003 report. Dr. Rosenberg testified that he determined that Claimant had some form of obturation abnormality to a disabling level, as well as some form of interstitial lung disease of a linear character with basilar predominance. He opined that the combination of Claimant's obturation abnormality, the potential interstitial lung disease, and the other medical problems that he had rendered Claimant incapable of performing his previous employments. Dr. Rosenberg stated that cigarette smoking can cause the types of changes evident on Claimant's chest x-rays. He commented that Claimant's lung volume testing was normal, at worst showing mild air flow obstruction based on the February 26, 2002 PFT. He noted that the diffusing capacity studies might be entirely normal after correcting for lung volumes. Dr. Rosenberg found that blood gas testing appears to demonstrate worsening oxygenation over time that has reached the point where Claimant is totally disabled based solely on the oxygenation impairment alone. He noted that the post-exercise ABG from May 4, 2001 was at a disabling level. Dr. Rosenberg noted that the study conducted on May 10, 1994, when Claimant was on 100% oxygen is very important for identifying the etiology of his oxygenation impairment. The use 100% oxygenation corrects all forms of abnormalities except for a shunt.

Since Claimant's May 10, 1994 test did not show improvement after administration of 100% oxygen, it shows that blood is being shunted from the right side of Claimant's heart to the left side of his heart in some fashion. Dr. Rosenberg could not identify with 100% accuracy what type of shunt was occurring in Claimant, other than to say that the May 10, 1994 study indicates that a physiologic shunt exists. He added that severe liver cirrhosis can cause shunting within the liver.

Dr. Rosenberg opined that the only problem, if any, that Claimant developed from his cigarette smoking history was that Claimant may have developed a mild degree of airflow obstruction. Dr. Rosenberg then restated his overview of Claimant's past medical history that revealed multiple other medical problems, including diabetes, peripheral vascular disease, coronary artery disease, history of alcoholism, reflux, and depression. He commented that esophageal reflux disease can cause chronic aspiration that is evident on a chest x-ray. He also noted that the immune response of liver disease can cause an inflammatory reaction in the lung causing linear changes that would appear on a chest x-ray. Dr. Rosenberg argued that the study relied upon by Dr. James in his report should not be relied upon by a prudent physician because the study did not control for cigarette smoking and coal dust exposure.

On cross-examination, Dr. Rosenberg admitted that the evidence is insufficient to establish the location of the shunt that he diagnosed. He also acknowledged that there is no medical evidence in the record that documents Claimant as suffering from any type of liver disease. Dr. Rosenberg noted that he did not actually diagnose the presence of chronic aspiration due to Claimant's reflux, he only testified that reflux can lead to chronic aspiration.

Dr. Renn was deposed on July 10, 2003. (EX 14). He reiterated the findings and conclusions contained in his June 5, 2003 report. Dr. Renn reviewed Claimant's Exhibits 1-4 prior to his deposition. He concluded that the information in Claimant's Exhibits 1-4 did not change his opinion regarding Claimant. Dr. Renn disagreed with the finding of the radiologist who interpreted the December 23, 1997 ventilation profusion scan as revealing COPD based on his determination that Claimant did not have COPD because his ventilatory function was normal and because repeated CT scans of Claimant's chest showed the absence of emphysema, bronchitis, and COPD. Instead, Dr. Renn found that the December 23, 1997 scan was further evidence that Claimant was suffering from a pulmonary embolism. Dr. Renn testified that the record contains evidence dating back to 1985 in support of a finding that Claimant was suffering from a pulmonary embolism. Dr. Renn added that Claimant's frequent immobilization during his numerous hospital stays and procedures placed Claimant at an increased risk for developing a pulmonary embolism. After considering Claimant's past medical history and symptoms and then comparing them with the risk factors identified in the Archives of Internal Medicine for developing a venous thromboembolic disease, Dr. Renn found that Claimant was at a very high risk level. Dr. Renn testified that Claimant's recurrent pulmonary emboli led to the development of a shunt mechanism by lodging in the small blood vessels that are responsible for picking up oxygen. When blood is foreclosed from obtaining oxygen in the alveolus due to the pulmonary emboli, the venous blood passes back to the heart and mixes with blood that has been oxygenated. This leads to desaturation. Dr. Renn opined that this form of shunt mechanism occurred in Claimant, causing Claimant's hypoxemia.

Dr. Renn testified that cigarette smoking can cause linear opacities to appear on chest x-rays. However, he personally does not believe that Claimant has any linear opacities as other physicians interpreting Claimant's chest x-rays have found. Dr. Renn noted that Claimant had gastroesophageal reflux disease ("GERD"). He opined, in reliance on a scientific medical article, that GERD will decrease a person's diffusing capacity in the range of 18-23%, which is significant. Dr. Renn opined that Claimant's reduction in diffusing capacity is more than adequately explained by the combination of Claimant's recurrent pulmonary emboli and his GERD.

Dr. Renn opined that the PFT conducted by Dr. James on May 4, 2001 was invalid because it was performed with poor cooperative effort as any knowledgeable pulmonologist would recognize. He also opined that the diffusing capacity study from February 26, 2002 was invalid based on his review of the graph tracings. Dr. Renn stated that the physicians who conducted the ABGs showing abnormal gas exchange should have suspected a pulmonary embolism because of the normal diffusing capacity, normal PFTs, and exercise-induced hypoxemia.

Dr. Renn identified a scientific medical article entitled "Idiopathic Pulmonary Fibrosis" published by the American College of Chest Physicians that identifies the criteria for diagnosing idiopathic pulmonary fibrosis absent a suitable surgical lung biopsy. In order to diagnose IPF, all three of the major criteria must be met in addition to at least three of the four minor criteria. The four major criteria are: (1) exclusion of other known causes of interstitial lung disease such as drug toxicities, environmental exposures, and connective tissue disease; (2) abnormal pulmonary function studies that include evidence of restriction; (3) bibasilar reticular abnormalities with minimal ground glass opacities on high resolution CT scans; and (4) transbronchial lung biopsy or bronchoalveolar lavage showing no features to support an alternative diagnosis. Dr. Renn stated that Claimant does not meet all of the major criteria because he does not have a restrictive defect, he doesn't have minimal ground glass opacities on the CT scan. He noted that the only criteria Claimant met was that he had environmental exposure. Dr. Renn stated that Claimant met three of the four minor criteria because he was over the age of 50, there was an insidious onset of otherwise unexplained dyspnea on exertion, and the duration of the illness was longer than three months. Since Claimant did not meet all of the major criteria, Dr. Renn concluded that it was foolish to even suspect that Claimant might have a pulmonary fibrosis.

Dr. Renn testified that he did not really have any comments regarding Dr. Potter's opinion because it merely contained his opinion and lacked any substantiating evidence. He disagreed with Dr. James' conclusion that the February 26, 2002 PFT showed evidence of an obstructive defect because the FEV1/FVC ration was normal for a man of Claimant's age. He credited Dr. James' statement that there was no consistent clinical evidence to show that Claimant had COPD or emphysema. Dr. Renn, reiterating his opinion that the February 26, 2002 diffusion capacity study was invalid, noted that Dr. James relied on the February 26, 2002 diffusion capacity study to conclude that Claimant's pneumoconiosis had worsened. Dr. Renn concluded by noting that Claimant was totally disabled due to chronic hypoxemia as a result of recurrent pulmonary emboli. He noted that Claimant's coal dust exposure was not a risk factor for developing a pulmonary emboli.

Dr. Farney was deposed on July 16, 2003. (EX 16). He reiterated the findings and conclusions contained in the report of his February 26, 2002 examination of Claimant. Dr. Farney acknowledged that GERD can cause chest x-ray abnormalities in the lower lung zones. He noted that he had reviewed the February 5, 1995 pulmonary angiogram that was positive for blood clots in Claimant's lungs. He referred to the pulmonary angiogram as the most definitive study for finding the presence or absence of blood clots in the lungs. Dr. Farney noted that Claimant had multiple risk factors for developing pulmonary emboli. He stated that a pulmonary embolism interferes with the matching of the ventilation and the profusion of the lungs, resulting in a ventilation profusion mismatch. Dr. Farney noted that such a mismatch causes hypoxia and a gas exchange impairment. He opined that the crackles he detected during his physical examination, which were mainly in the left lung base, may be due to fluid in Claimant's lungs or fibrosis in the lower lungs. Dr. Farney's impression of the ABG that he conducted on February 26, 2002 was that it was normal for Claimant's age and elevation. He opined that the PFT he conducted on February 26, 2002 showed that Claimant's lung volumes were normal and that he did not have a restrictive lung disease. However, he did note that the lung volumes show some evidence of deterioration since the PFT he conducted on December 23, 1997. Dr. Farney opined that the most likely causes of Claimant's low diffusion capacity were his history of pulmonary embolism and his heavy cigarette smoking history that may have led to some degree of emphysema.

Dr. Farney criticized Dr. James' opinion finding the presence of pulmonary fibrosis because Dr. Farney did not think that Claimant had pulmonary fibrosis because the last chest x-ray and most recent CT scan did not show fibrosis. He added that the only evidence of fibrosis were a few chest x-rays variously interpreted as showing minimal fibrosis inconsistently. Dr. Farney concluded by testifying that Claimant had pulmonary disease in the form of a history of pulmonary embolism, probably sleep apnea, and a suspicion of small airways disease and some emphysema.

Dr. Repsher was deposed on September 29, 2003. (EX 17). He testified that previously reviewed Claimant's medical records in 1998, issued a consultative report on May 7, 1998, and testified at the formal hearing on June 22, 1998. In addition to reconsidering the prior records, Dr. Repsher also reviewed Employer's Exhibits 1-13, Claimant's Exhibits 1-4, the 1995 pulmonary angiogram, and the most recent reports of Drs. Farney, Potter, James, as well as the deposition of Dr. Renn. Dr. Repsher testified that he did not believe that Claimant had a material change in his respiratory or pulmonary condition from 1998 until the date of the deposition. He noted that a pulmonary angiogram is a test conducted to diagnose or rule out pulmonary emboli. Dr. Repsher stated that the 1995 pulmonary angiogram revealed at least two pulmonary emboli. He described the problem that a pulmonary emboli causes as blocking the blood flow through a particular blood vessel, which leads to a series of events involving physiologic shunting that may cause pulmonary infarction or death of lung tissue. Dr. Repsher testified that this process very commonly causes the person to become hypoxemic, a situation that usually resolves with the resolution of the clot. He noted that Claimant has had varying hypoxemia and varying amounts of shunts determined (from 6-7% on one occasion to 17% on another and then back to 6% on another). Dr. Repsher referred to the scientific medical article that Dr. Renn referenced in his deposition testimony regarding the risk factors for developing thromboembolic disease. He testified that Claimant had GERD, which he noted can cause a large variety of pulmonary

problems from aggravating asthma to recurrent aspiration pneumonia with pulmonary fibrosis of the lower lobes to causing chronic bronchitis or asthma. Dr. Repsher also noted that Claimant had a carbon monoxide diffusion capacity problem. He acknowledged another scientific medical article referenced by Dr. Renn in his deposition that found an association between GERD and a reduced carbon monoxide diffusing capacity problem.

Dr. Repsher testified that he found no biopsy evidence of pneumoconiosis, adding that the most recent chest x-ray evidence was negative. He added that when previous chest x-rays that were positive for pneumoconiosis are followed by chest x-rays that are negative for pneumoconiosis, it strongly suggests that whatever was on the x-ray interpreted as positive was not CWP since CWP does not ever improve. From his review of the records, Dr. Repsher testified that Claimant may have very mild, clinically insignificant COPD. However, he agreed with Dr. James that there was no clear cut evidence of other COPD or emphysema. Dr. Repsher disagreed with Dr. James discussion of the chest x-ray evidence. He stated that Dr. James made up a heretofore nonexistent disease that apparently only Dr. James recognizes when he diagnosed lower lobe fibrotic CWP. Dr. Repsher testified that lower lobe fibrotic CWP has never been described pathologically or radiographically, and it has not been documented by scientific medical articles. He noted the articles that Dr. James cited to, but criticized those articles for neglecting to recognize the fact that lower lobe linear opacities are related to cigarette smoking or for failing to account for cigarette smoking.

Dr. Repsher testified that Claimant does not have coal mine induced lung disease because he had no radiographic evidence of CWP and he does not have clinically significant COPD. In response to the question asking what was wrong with Claimant's lungs, Dr. Repsher answered, "[n]ot much." Again, he noted that Claimant might have mild COPD that was statistically related to his previous smoking habit, but that it was not clinically significant. He opined that Claimant's respiratory symptoms of shortness of breath are related to his coronary artery disease combined with a sedentary lifestyle and significant obesity. Dr. Repsher commented that diffuse pulmonary fibrosis is not the same diagnosis as CWP according to the terms' common use in medicine. He noted that diffuse pulmonary fibrosis usually refers to interstitial lung diseases that produce linear opacities and not rounded opacities. From a pulmonary standpoint, Dr. Repsher opined that Claimant could perform his previous coal mine employment. However, from a cardiac and general fitness standpoint, Claimant would probably not be able to perform his previous coal mine employment. He concluded that Claimant did not have a totally disabling respiratory impairment of his respiratory parenchyma. Dr. Repsher allowed that Claimant might have some element of chronic thromboembolic pulmonary hypertension, but that would be in the respiratory blood vessels and not in the respiratory tissue itself. Dr. Repsher concluded that Claimant's smoking history of 25-50 pack years may have caused some mild COPD and possibly some reduction in his diffusing capacity.

Hospital Records

On April 22, 1998, Douglas Wing, M.D. conducted a abdominal aortogram and peripheral run off study, which was followed by a balloon angioplasty through Claimant's right superficial femoral artery at the Utah Valley Regional Medical Center. (EX 6). After the angioplasty, Claimant reported that the burning sensation in his right foot began to improve.

Claimant's cardiologist, John Frischknecht, M.D., conducted a left heart catheterization with left ventricular and coronary angiograms on September 22, 1998, which revealed significant disease in Claimant's right coronary artery and circumflex. (EX 6). He determined that it was amenable to percutaneous transluminal coronary angioplasty ("PTCA"). After Claimant agreed, Dr. Frischknecht conducted a PTCA and stent placement of the right coronary artery and circumflex.

Dr. Frischknecht examined Claimant on October 21, 1998. (EX 6). Claimant presented for a follow-up regarding his coronary artery disease, after recently having a PTCA and stent. Claimant reported no symptoms of heart failure or angina, and he had been able to exercise some. Claimant's chest was clear on physical examination. Dr. Frischknecht's assessment was coronary artery disease that was stable without evidence of angina and stable gastroesophageal reflux.

Claimant was treated on November 24, 1998 at Castleview Hospital by Dr. Brian Peterson. (EX 5). Claimant had experienced a nosebleed two weeks ago that Dr. Peterson treated, and he returned with another nosebleed. Dr. Peterson diagnosed epistaxis and admitted Claimant overnight.

Dr. Potter referred Claimant to Dr. Frischknecht at the Utah Valley Regional Medical Center on February 26, 1999. Dr. Frischknecht noted that Claimant had a coronary artery disease and had recently undergone a PTCA and stent two months ago. Claimant began experiencing chest pain one week prior to the hospital admission. Claimant's chest was clear on physical examination. Dr. Frischknecht performed a left heart catheterization with left ventricular and coronary angiograms. His assessment was that Claimant had a high-grade lesion in the circumflex coronary artery that was amenable to interventional procedures. Thus, Dr. Frischknecht submitted Claimant to another PTCA. His discharge diagnosis was progressive angina and two-vessel coronary artery disease.

On January 24, 2000, Claimant underwent an adenosine myocardial perfusion imaging, that was conducted by Dr. Frischknecht, to define the likelihood of stress-induced ischemia and to assess Claimant's cardiac prognosis due to his atypical angina, previous PTCA, and previous coronary stenting. (EX 6). His overall impression from the procedure was that there is a low intermediate likelihood of significant stress-induced ischemia. Dr. Frischknecht found that the location and type of perfusion defect may be consistent with very minimal ischemia in the distal inferolateral segment. However, he noted that the small size and mild degree of the defect limit diagnostic certainty. Claimant's left ventricular size and systolic function were normal at rest.

On May 21, 2001, Claimant was admitted to Utah Valley Regional Medical Center by Dr. Frischknecht for a PTCA of his right coronary artery. Dr. Frischknecht performed another PTCA and stent placement of the right coronary artery. His discharge diagnoses were progressive angina and single-vessel coronary artery disease.

Dr. Frischknecth examined Claimant on July 12, 2001. (EX 6). His pre-examination problem list noted coronary artery disease, hypercholesterolemia, and diabetes. Claimant had no complaints of chest pain or shortness of breath and reported getting along well. His lungs were clear to auscultation. Dr. Frischknecht assessed Claimant as stable and planned to continue Claimant's current medications.

Claimant presented to the Utah Valley Regional Medical Center on November 21, 2001 because his nose was bleeding. (EX 6). Dr. Brian Hyer examined Claimant, diagnosed epistaxis, applied a Mercocel pack and discharged Claimant when there was no active bleeding.

Other Medical Evidence

Dr. Howard Mann interpreted the CT scan obtained by Dr. Farney on February 26, 2002. He detected subsegmental opacities in the lower lung zones, typical of atelectasis. Dr. Mann did not see any findings consistent with emphysema, interstitial fibrosis, or simple/complicated inorganic dust pneumoconiosis. He found dependent lung opacities consistent with underexpanded lung. Dr. Mann also found that there were many mediastinal and bilateral hilar lymph nodes, most of which were 1 cm of less in diameter. Finally, Dr. Mann detected calcifications in the aortic valve and left main coronary artery.

Jerome Wiot, M.D., who is a board-certified radiologist and B-reader, interpreted a CT scan obtained on February 26, 2002 and summarized his findings in a report dated May 29, 2002. (DX 76). He opined that the CT scan shows no evidence of CWP. He did find minimal basilar septal thickening and interstitial change of unknown etiology that was not related to coal dust exposure. He found no pleural plaques to suggest asbestos exposure. Dr. Wiot noted that there are multiple causes of basilar interstitial fibrosis, the most common of which is idiopathic pulmonary fibrosis.

In a report dated June 26, 2002, Ralph Shipley, M.D., who is dually board-certified as a radiologist and B-reader, interpreted the CT scan obtained on February 26, 2002. (DX 85). Dr. Shipley detected focal opacities in both lower lung lobes, greater on the right and primarily linear and irregular. He also detected calcification in the coronary arteries that indicate atherosclerosis. His impression was that the lower lobe opacities that he detected were likely post-inflammatory scarring, perhaps from old pneumonia. Dr. Shipley concluded that he did not find any pleural or parenchymal evidence of pneumoconiosis.

Dr. Spitz, who is dually board-certified as a radiologist and B-reader, interpreted the CT scan obtained on February 26, 2002 in a report dated August 3, 2002. (DX 86). He noted that the CT scan shows no evidence of nodules, large masses, or eggshell calcifications. He detected evidence in the lung windows of basilar interstitial disease with septal thickening. Dr. Spitz did not find any evidence of pleural plaques. His impression was that there was no evidence of CWP

and that there were findings consistent with basilar interstitial fibrosis, possibly representing IPF.[8]

Smoking History

Claimant testified at the hearing that he smoked one pack of cigarettes per day for 26 years, and that he last smoked in 1978. (Tr. 46). In the interrogatories propounded to Claimant on March 19, 2001, Claimant stated that he began smoking at the age of 19 and quit smoking in 1978. He noted that he smoked approximately one pack of cigarettes per day. He also disputed Dr. Lincoln's account that he smoked three packs of cigarettes per at one time. (Tr. 47). Thus, according to Claimant's account, his smoking history amounts to 26 pack-years. Claimant's account of his smoking history is credible. Therefore, I find that Claimant's smoked cigarettes for 26 pack-years.

DISCUSSION AND APPLICABLE LAW

Mr. Byrge's claim was made after March 31, 1980, the effective date of Part 718, and must therefore be adjudicated under those regulations. To establish entitlement to benefits under Part 718, Claimant must establish, by a preponderance of the evidence, that he:

1. Is a miner as defined in this section; and

2. Has met the requirements for entitlement to benefits by establishing that he:

   (i) Has pneumoconiosis (see § 718.202), and

   (ii) The pneumoconiosis arose out of coal mine employment (see § 718.203), and

   (iii) Is totally disabled (see § 718.204(c)), and

   (iv) The pneumoconiosis contributes to the total disability (see § 718.204(c)); and

3. Has filed a claim for benefits in accordance with the provisions of this part.

Section 725.202(d)(1-3); *see also* §§ 718.202, 718.203, and 718.204(c).

Modification

Section 22 of the Longshore and Harbor Workers' Compensation Act, 33 U.S.C. § 922, as incorporated into the Black Lung Benefits Act by 30 U.S.C. § 932(a) and as implemented by § 725.310, provides that upon his or her own initiative, or upon the request of any party on the

[8] Dr. Spitz's report does not expressly define whether his impression of IPF refers to idiopathic pulmonary fibrosis or interstitial pulmonary fibrosis. The abbreviation of "IPF" is defined by the publication *Medical Abbreviations*, Neil M. Davis (6th Edition) as "idiopathic pulmonary fibrosis." From the context of Dr. Spitz's report and the publication's idenfitication, I find that Dr. Spitz's impression of IPF is an impression of idiopathic pulmonary fibrosis.

ground of a change in conditions or because of a mistake in a determination of fact, the deputy commissioner may, at any time prior to one year after the date of the last payment of benefits, or at any time before one year after the denial of a claim, reconsider the terms of an award or a denial of benefits. § 725.310(a).

In deciding whether a mistake in fact has occurred, the United States Supreme Court stated that the Administrative Law Judge has broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted. *O'Keefe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 256 (1971). Regardless of whether a specific allegation is made, the administrative law judge is obligated to review the entire record to determine if a mistake in determination of any fact occurred. *Kingery v. Hunt Branch Coal Co.*, 19 B.L.R. 1-6 (1994).

In determining whether a change in conditions has occurred requiring modification of the prior denial, the Benefits Review Board ("Board") stated that,

> the Administrative Law Judge is obligated to perform an independent assessment of the newly submitted evidence (all evidence submitted subsequent to the prior denial), considered in conjunction with the previously submitted evidence, to determine if the weight of the new evidence is sufficient to establish at least one element of entitlement which defeated entitlement in the prior decision.

Kingery, 19 B.L.R. 1-6; *See also Napier v. Director, OWCP*, 17 B.L.R. 1-111 (1993); *Nataloni v. Director, OWCP*, 17 B.L.R. 1-82 (1993). Furthermore,

> if the newly submitted evidence is sufficient to establish modification . . ., the Administrative Law Judge must consider all of the evidence of record to determine whether Claimant has established entitlement to benefits on the merits of the claim.

Kovac v. BNCR Mining Corp., 14 B.L.R. 1-156 (1990), *modified on recon.*, 16 B.L.R. 1-71 (1992).

Mistake in a Determination of a Fact

The Claimant has not alleged that any mistake of fact was committed in the prior denial of benefits. I have reviewed the decision and all of the evidence before Administrative Law Judge Mosser for consideration. I found no mistake in any determination of fact in the prior decision and order denying benefits that warrants a *de novo* review of the entire record. However, Claimant's request for modification may still be granted if the newly submitted evidence establishes a change in conditions.

Change in Conditions

The previous decision and order, from which Claimant seeks modification, denied benefits to Claimant after it was determined that he had not established the existence of

pneumoconiosis. Therefore, Claimant may demonstrate a change in conditions by establishing the presence of pneumoconiosis, which was the element of entitlement previously adjudicated against him.

Pneumoconiosis

Claimant may establish a material change in conditions by proving the existence of pneumoconiosis under § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. *See Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994). Pneumoconiosis is defined by the regulations:

> (a) For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical" pneumoconiosis and statutory, or "legal" pneumoconiosis.
>
> (1) *Clinical Pneumoconiosis*. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.
>
> (2) *Legal Pneumoconiosis*. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.
>
> (b) For the purposes of this section, a disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment.
>
> (c) For purposes of this definition, "pneumoconiosis" is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure.

Sections 718.201(a-c).

Section 718.202(a) sets forth four methods for determining the existence of pneumoconiosis.

(1) Under § 718.202(a)(1), a finding that pneumoconiosis exists may be based upon x-ray evidence. The newly submitted evidentiary record contains 12 interpretations of three chest x-rays obtained since the prior denial of benefits, two interpretations rendered after the prior denial from a film that was obtained before the prior denial, and one quality-only interpretation. Dr. James, who is a B-reader, interpreted a film dated May 15, 2000 as positive for pneumoconiosis. There were no interpretations to the contrary. Therefore, I find that the May 15, 200 film is positive for pneumoconiosis. Dr. James also interpreted a film obtained on May 4, 2001 as positive for pneumoconiosis. In contrast, Drs. Wiot, Spitz, Shipley, and Preger, all of whom are dually-certified as radiologists and B-readers, interpreted the film as negative for pneumoconiosis. I accord greater probative weight to the more numerous negative interpretations that were rendered by better-qualified physicians. Therefore, I find that the May 4, 2001 film is negative. Drs. Wiot, Shipley, and Spitz, all of whom are dually-certified physicians, interpreted the February 26, 2002 film as negative. Two B-readers, Drs. James and Renn, interpreted the February 26, 2002 film as negative. Dr. Mann, who does not hold any advanced credentials for interpreting chest x-rays, also found the February 26, 2002 film to be negative. All six physicians interpreted the February 26, 2002 film as negative for the existence of pneumoconiosis. There were no positive interpretations. Therefore, I find that the February 26, 2002 x-ray is negative for the existence of pneumoconiosis. Of the 12 newly submitted chest x-ray interpretations, 10 were negative for the existence of pneumoconiosis. I determined that the two most recent both newly submitted chest x-rays were negative. Therefore, I find that Claimant has failed to establish the presence of pneumoconiosis under subsection (a)(1).

(2) Under § 718.202(a)(2), a determination that pneumoconiosis is present may be based, in the case of a living miner, upon biopsy evidence. The evidentiary record does not contain any newly submitted biopsy evidence. Therefore, I find that the Claimant has failed to establish the existence of pneumoconiosis through biopsy evidence under subsection (a)(2).

(3) Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found to be applicable. In this case, the presumption of § 718.304 does not apply because there is no evidence in the record of complicated pneumoconiosis. Section 718.305 is not applicable to claims filed after January 1, 1982. Finally, the presumption of § 718.306 is applicable only in a survivor's claim filed prior to June 30, 1982. Therefore, Claimant cannot establish pneumoconiosis under subsection (a)(3).

(4) The fourth and final way in which it is possible to establish the existence of pneumoconiosis under § 718.202 is set forth in subsection (a)(4) which provides in pertinent part:

> A determination of the existence of pneumoconiosis may also be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers or suffered from pneumoconiosis as defined in § 718.201. Any such finding shall be based on electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

§ 718.202(a)(4).

This section requires a weighing of all relevant medical evidence to ascertain whether or not the claimant has established the presence of pneumoconiosis by a preponderance of the evidence. Any finding of pneumoconiosis under § 718.202(a)(4) must be based upon objective medical evidence and also be supported by a reasoned medical opinion. A reasoned opinion is one which contains underlying documentation adequate to support the physician's conclusions. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). Proper documentation exists where the physician sets forth the clinical findings, observations, facts, and other data on which he bases his diagnosis. *Oggero v. Director, OWCP*, 7 B.L.R. 1-860 (1985).

Dr. Potter issued a narrative medical opinion on October 25, 1999, wherein he found that Claimant had a pulmonary disease related to his coal mine employment. He also opined that Claimant's lung disease is related to and worsened by his 30 years of working at the face of the coal mine. This opinion is not reasoned or documented. Dr. Potter did not identify any clinical observations and findings, nor did he rely upon adequate data to support his conclusion. Dr. Potter issued a second narrative opinion on December 4, 2000. He found that Claimant continued to have COPD that was made worse by Claimant's 30 year history of working mostly at the face of the coal mine. In his December 2000 opinion, Dr. Potter also found that Claimant's coal dust exposure was a significant factor in Claimant's lung disease and his continued need for supplemental oxygen. He attempted to bolster his opinion by noting that Claimant had not smoked for 22 years. However, aside from noting Claimant's smoking and coal mine employment history, Dr. Potter failed to identify any clinical observations or findings to support his conclusions. Dr. Potter's December 2000 opinion is not reasoned or documented. Dr. Potter offered his third narrative opinion on June 16, 2003. He noted that he had been treating Claimant since 1988 for a progressive lung disease that requires supplemental oxygen. He opined that Claimant's coal dust exposure and smoking history contributed to his pulmonary disability. Dr. Potter commented that a CT scan showed pulmonary fibrosis, and he pointed to Claimant's demonstrated dyspnea on exertion and hypoxemia. Based on Claimant's pulse oximetry and ABGs, Dr. Potter stated that Claimant is disabled due to his lungs. Dr. Potter concluded that the identified chest x-ray finding of pulmonary fibrosis was significantly caused by Claimant's smoking and coal dust exposure. He also opined that Claimant's condition had deteriorated over the past years after comparing a 1994 PFT to a 2002 PFT. Dr. Potter then concluded that Claimant suffered from simple CWP based on his 14 years of treating Claimant, his review of the chest x-rays, PFTs, and ABGs. Dr. Potter set forth clinical observations and findings, and he relied upon adequate date to support his conclusion. He considered an accurate account of Claimant's smoking and coal mine employment history, physically examined Claimant, and interpreted the results of objective testing Claimant underwent. His June 16, 2003 opinion is reasoned and documented. Therefore, I find that Dr. Potter's June 16, 2003 opinion is entitled to probative weight.

In determining the weight to be accorded to a treating physician's opinion, the amended regulations at § 718.104(d) (2002) are not directly applicable because this evidence was developed prior to January 19, 2001; but it is instructive. *See Wolf Creek Collieries v. Director, OWCP [Stephens]*, 298 F.3d 511 (6th Cir. 2002). An administrative law judge may rely upon the well-reasoned and well-documented opinion of a treating physician as substantial evidence in

awarding that physician's opinion controlling weight based upon four factors: (1) nature of relationship; (2) duration of relationship; (3) frequency of treatment; and (4) extent of treatment. § 718.104(d) (2002). Dr. Potter noted that he had treated Claimant's pulmonary disease for 14 years before issuing his June 16, 2003 report. It appears that Dr. Potter prescribed supplemental oxygen to treat Claimant's pulmonary impairment. Overall, the record indicates that Dr. Potter treated Claimant for a lengthy period for a pulmonary impairment. However, the record simply does not establish that Dr. Potter obtained superior and relevant information regarding Claimant's condition based on their treating relationship. The evidence identified by Dr. Potter was available for review by the other physicians who rendered opinions in this case. Beyond Dr. Potter's comments regarding Claimant's demonstrated dyspnea on exertions and hypoxemia, which aren't necessarily his own personal observations, Dr. Potter's report does not reference any of his own clinical findings or observations derived from personally examining Claimant. While I find that Dr. Potter's conclusion is relevant and probative, I decline to grant controlling weight to his opinion due to his status as Claimant's treating physician.

Dr. James examined Claimant on May 4, 2001 and issued a narrative opinion. He concluded that Claimant suffered from a diffuse lung disease most prominent in the lower lung zones secondary to the chronic inhalation of coal mine dust. Dr. James added that CWP can be present with more prominent lower lobe interstitial disease instead of the more commonly found upper lobe, rounded opacities of CWP. He interpreted a chest x-ray as positive for pneumoconiosis, and he referenced a 1997 CT scan and the transbronchial biopsy that he found to reveal pulmonary fibrosis. Dr. James detected decreased breath sounds during examination of Claimant's chest. He ruled out exposure to asbestos as a potential cause of the pulmonary fibrosis. Dr. James considered an accurate account of Claimant's smoking and coal mine employment histories. He set forth clinical observations and findings, and his reasoning is supported by adequate data. This opinion is reasoned and documented.

Dr. James issued a second narrative medical opinion more than two years later on June 25, 2003. He concluded that the pulmonary function and oxygenation abnormalities are more likely than not secondary to the diffuse lung disease that is observed on the radiographic studies and are due to the chronic inhalation of coal mine dust. Dr. James noted that chest x-rays interpreted by other physicians, as well as his own interpretation, detected the presence of abnormalities consistent with pneumoconiosis even though other physicians interpreted the x-rays as negative. He also identified a 1994 CT scan that noted minimal interstitial densities in Claimant's lower lung bases. Dr. James found that there was no consistent clinical evidence to find that Claimant suffered from emphysema or COPD. He noted that the only time an airflow obstruction was detected was in the February 26, 2002 PFT. Dr. James further supported this finding by noting that the CT scans from 1994, 1997, and 2002 did not show evidence of emphysema. Dr. James found it to be more likely than not that Claimant developed diffuse fibrotic lung disease from his long-term exposure to coal mine dust. He identified how a fibrotic process develops in the lungs of hard rock coal miners in comparison to the usual manner in which CWP develops. Dr. James added that cigarette smoking may be a contributing factor to the development of fibrotic lung disease. He referenced scientific medical articles to support his conclusions. He ruled out any other non-coal mine dust induced abnormality as the reason for Claimant's radiographic and physiologic abnormalities. Dr. James found it less likely that Claimant had COPD, but stated that additional testing would determine whether the airflow

limitation detected in the February 2002 PFT is persistent and not due to an acute process like bronchitis. He found that the radiographic abnormalities were not consistent with a diagnosis of COPD, and he ruled out a diagnosis of idiopathic pulmonary fibrosis because Claimant did not demonstrate the usual findings of crackles and clubbing of digits associated with idiopathic pulmonary fibrosis. He also ruled out sarcoidosis and asbestosis. Dr. James found it to be unlikely that Claimant's coronary artery disease caused the pulmonary fibrosis because Claimant didn't have findings of congestive heart failure, which shows an association between coronary artery disease and pulmonary fibrosis. Dr. James commented that, even though Claimant is obese and obesity can alter lung function by decreasing vital capacity, he noted that obesity does not alter diffusion capacity. He acknowledged records indicating that Claimant underwent a shunt study and findings of a 6% and 17% shunt. Dr. James stated that a shunt occurs in 5% of all healthy people. However, he added that Claimant has no evidence of the causes of a shunt, which include congenital heart disease, lung cancer, pulmonary arteriovenous shunts, or shunts due to liver disease. Dr. James further noted that the CT scans did not show evidence of a shunt. He based his June 2003 opinion on his May 4, 2001 examination and his review of Claimant's medical records. Dr. James set forth clinical observations and findings, and his reasoning is supported by adequate data. He considered an accurate account of Claimant's smoking and coal mine employment histories. Dr. James relied upon scientific medical literature. His opinion is reasoned and documented. I find that Dr. James' 2001 and 2003 opinions are entitled to probative weight enhanced by his credentials as a board-certified pulmonologist.

Dr. Farney issued a narrative opinion on February 26, 2002. He opined that Claimant did not have CWP or any other pulmonary disease due to coal dust exposure. Dr. Farney noted that Claimant had a reduction in diffusing capacity, but he attributed it to emphysema associated with Claimant's smoking history. Dr. Farney noted that Dr. Mann interpreted a chest x-ray and CT scan obtained on February 26, 2002 as negative for CWP or fibrosis. Dr. Farney reiterated these opinions during his deposition testimony on July 16, 2003. He noted that GERD can cause chest x-ray abnormalities in the lower lung zones. He testified that the PFT and ABG conducted on February 26, 2002 were normal for Claimant's age and elevation. Dr. Farney also discussed the findings of a pulmonary embolism from the February 1995 pulmonary angiogram. He found that Claimant had multiple risk factors for developing pulmonary emboli. Dr. Farney testified that a pulmonary embolism interferes with the matching of the ventilation and profusion of the lungs, which can cause hypoxia and a gas exchange impairment. He attributed the crackles he detected during physical examination of Claimant's lungs to fluid or fibrosis in the lungs. Dr. Farney concluded that the most likely causes of Claimant's low diffusion capacity were his history of pulmonary emboli and his heavy cigarette smoking history that may have led to some degree of emphysema. He also criticized Dr. James' conclusion that Claimant had pulmonary fibrosis since the most recent chest x-ray and CT scan did not show evidence of pulmonary fibrosis. He reviewed his examination report from his December 23, 1997 exam of Claimant, and he considered an accurate account of Claimant's smoking and coal mine employment histories. Dr. Farney set forth clinical observations and findings, and his reasoning is supported by adequate data. His opinion is reasoned and documented. I find that Dr. Farney's opinion is entitled to probative weight.

Dr. Rosenberg issued a consultative report on April 28, 2003 after reviewing Claimant's medical records. He opined that the chest x-ray findings of basilar linear opacities are inconsistent with the upper lobe micronodular changes associated with the past inhalation of coal dust. Dr. Rosenberg opined that the pattern of the linear opacities is not consistent with an interstitial lung disease arising out of coal mine employment. Instead, he noted that the type of development can occur consequent to cigarette smoking. Thus, Dr. Rosenberg expressly ruled out the interstitial form of CWP. Dr. Rosenberg found that Claimant did not suffer from any pulmonary restriction, but he did find that Claimant had an increasing gas exchange abnormality with exercise. He found that Claimant appears to have a right to left shunt, which he stated was not related to coal dust exposure. He also opined that any minimal degree of obstructive lung disease that Claimant suffers from is not related to coal dust exposure because there is no evidence of micronodularity. He attributed the minimal degree of obstructive lung disease to Claimant's smoking history. During his July 8, 2003 deposition, Dr. Rosenberg reiterated the findings and conclusions contained in his April 28, 2003 report. He supported his conclusion that Claimant's apparent shunt is unrelated to coal dust exposure because the shunt appeared during a test obtained while Claimant was on 100% oxygen, since 100% oxygen corrects all forms of abnormalities except for a shunt. Dr. Rosenberg testified that the minimal degree of COPD evident from the February 26, 2002 PFT was solely due to Claimant's smoking history. Dr. Rosenberg identified other potential etiologies for the linear opacities that appear on Claimant's chest x-rays, including GERD that causes chronic aspiration and an immune response to liver disease. He stated that he would not rely upon the scientific medical articles cited by Dr. James since the underlying studies did not control for smoking and coal dust exposure. However, Dr. James noted that he can not establish the location of the shunt, and that there were no actual diagnoses of liver disease or chronic aspiration due to GERD. Dr. Rosenberg set forth clinical observations and findings, and his reasoning is supported by adequate data. He considered an accurate account of Claimant's smoking and coal dust exposure histories. Dr. Rosenberg relied upon scientific medical articles to support his conclusions. His opinion is reasoned and documented. I find that Dr. Rosenberg's opinion is entitled to probative weight enhanced by his credentials as a board-certified pulmonologist.

Dr. Renn issued a consultative report on June 9, 2003. He opined that Claimant had recurrent hypoxemia caused by pulmonary emboli. Dr. Renn found that Claimant suffered from mild resting hypoxemia that had generally persisted, as well as from exercise-induced hypoxemia. Based on the ABG conducted while Claimant was on 100% oxygen, Dr. Renn concluded that Claimant's alveolar-arterial oxygen gradient indicated a fairly marked shunt. Dr. Renn also found that the 1997 and 2002 CT scans did not show evidence of CWP; there was evidence of minimal bibasilar interstitial fibrotic changes. During his July 10, 2003 deposition, Dr. Renn reiterated the findings and conclusions contained in his June 9, 2003 report. He disagreed with the interpretation of the December 1997 ventilation profusion scan that found COPD because Dr. Renn considered Claimant's ventilatory function to be normal and because repeated CT scans of Claimant's chest showed the absence of emphysema, bronchitis, and COPD. Rather, he stated that the December 1997 scan was further evidence that Claimant suffered from a pulmonary embolism that dated back to 1985. He identified the risk factors Claimant had that led Dr. Renn to conclude that Claimant was at a high risk for developing pulmonary emboli. He explained the physiologic process through which a shunt leads to hypoxemia. Dr. Renn did not believe that Claimant had any linear opacities in his lungs, but he

did note that cigarette smoking can cause linear opacities. Dr. Renn then cited to a scientific medical article that found that GERD can decrease a person's diffusing capacity by 18-23%. Thus, Dr. Renn concluded that the reduction in Claimant's diffusing capacity was easily explained by his recurrent pulmonary emboli and GERD. Dr. Renn then addressed Dr. James' diagnosis of pulmonary fibrosis. He identified the criteria established by the American College of Chest Physicians for diagnosing pulmonary fibrosis. Dr. Renn concluded that Claimant does not meet all of the major criteria for diagnosis pulmonary fibrosis because he does not suffer from a restrictive defect and because there is no evidence of minimal ground glass opacities on the CT scans. He agreed with Dr. James' finding that there was no evidence to show that Claimant had COPD or emphysema. He also testified that he did not have any comments for Dr. Potter's opinion because his opinion lacked substantiation. Dr. Renn set forth clinical observations and findings, and his reasoning is supported by adequate data. He considered an accurate account of Claimant's smoking and coal mine employment histories. He substantiated his opinion by relying upon scientific medical articles. His opinion is reasoned and documented. I find that Dr. Renn's opinion is entitled to probative weight enhanced by his credentials as a board-certified pulmonologist.

Dr. Tuteur issued a consultative report on June 16, 2003 based on his review of Claimant's medical records and his examination of Claimant in 1998. He opined that Claimant did not have CWP or any other coal mine dust-induced disease process that is of sufficient severity and profusion to produce clinical symptoms, physical examination abnormalities, impairment of pulmonary or respiratory function, or radiographic abnormalities. Dr. Tuteur noted that Claimant had developed multiple pulmonary emboli and right to left shunting. He opined that the newly submitted radiographic evidence continued to be absent of changes compatible with CWP. Dr. Tuteur concluded that Claimant's impairment of respiratory function and gas exchange abnormalities were due to his coronary artery disease and the presence of a right to left shunt. He could not identify the physiologic location of the shunt. Dr. Tuteur set forth clinical observations and findings, and his reasoning is supported by adequate data. He considered an accurate account of Claimant's smoking and coal mine employment histories. His opinion is reasoned and documented. I find that Dr. Tuteur's opinion is entitled to probative weight enhanced by his credentials as a board-certified pulmonologist.

Dr. Repsher was deposed on September 29, 2003. He noted that he had previously issued a consultative report in 1998 after reviewing Claimant's medical records, testified at the previous hearing, and reviewed the newly submitted medical evidence. Dr. Repsher noted that the 1995 pulmonary angiogram identified at least two pulmonary emboli. He noted that pulmonary emboli lead to blocked blood flow, which leads to a series of events involving shunting and may lead to hypoxemia or pulmonary infarction and death of lung tissue. Dr. Repsher reference the scientific medical article that Dr. Renn relied upon to conclude that Claimant was at a high risk for developing pulmonary emboli. He also referenced the article that Dr. Renn relied upon to establish the connection between GERD and linear opacities evident on chest x-ray. Dr. Repsher also relied upon an article to establish a link between Claimant's GERD and his reduced diffusing capacity. Dr. Repsher found no biopsy evidence of CWP, and he found the newly submitted chest x-ray evidence to be negative for CWP. He allowed that Claimant might have mild, clinically insignificant COPD, but he agreed with Dr. James' opinion that there is no clear cut evidence to diagnose other COPD or emphysema. Dr. Repsher then testified that Dr. James'

diagnosis of lower lobe fibrotic CWP amounted to making up a heretofore nonexistent disease that only Dr. James' recognizes. He stated that lower lobe fibrotic CWP has never been described pathologically or radiographically, and it has never been documented by scientific medical articles. He criticized the articles that Dr. James' relied upon for neglecting to recognize the fact that lower lobe linear opacities are related to cigarette smoking or for failing to account for cigarette smoking. Dr. Repsher testified that, besides the potentially mild degree of COPD, there was not much wrong with Claimant's lungs. He attributed Claimant's shortness of breath to his coronary artery disease, sedentary lifestyle, and significant obesity. Dr. Repsher set forth clinical observations and findings, and his reasoning is supported by adequate data. He considered an accurate account of Claimant's smoking and coal mine employment histories. Dr. Repsher's conclusions are supported by scientific medical literature. His opinion is reasoned and documented. I find that Dr. Repsher's opinion is entitled to probative weight enhanced by his credentials as a board-certified pulmonologist.

Dr. Mann interpreted the February 26, 2002 CT scan, finding subsegmental opacities in the lower lung zones typical of atelectasis. Dr. Wiot interpreted the February 26, 2002 CT scan as negative for CWP. He did find minimal basilar septal thickening and interstitial change of unknown etiology. Dr. Wiot opined that there are numerous causes of basilar interstitial fibrosis, including idiopathic pulmonary fibrosis. Dr. Shipley detected the presence of focal opacities in both lower lung lobes that were primarily linear and irregular in the February 26, 2002 CT scan. He attributed the lower lobe opacities to post-inflammatory scarring, perhaps from an old pneumonia. Lastly, Dr. Spitz interpreted the February 26, 2002 CT scan as containing evidence in the lung windows of basilar interstitial disease with septal thickening. He opined that there was no evidence of CWP, adding that the basilar interstitial fibrosis could be IPF.

The hospital records document Claimant undergoing repeated PTCAs with stent placements dating back to 1998. Dr. Frischknecht found Claimant to be suffering from significant coronary artery disease and stable GERD in 1998.

The newly submitted evidentiary record contains two reasoned narrative medical opinions finding that Claimant suffered from pneumoconiosis, five physicians who found that Claimant did not suffer from CWP, a CT scan revealing an interstitial lower lung lobe disease of unknown etiology that was not consistent with CWP, and hospital records establishing a history of repeated PTCAs with stent placement, coronary artery disease, and stable GERD. I find that the newly submitted narrative medical evidence is insufficient to establish the presence of pneumoconiosis.

Dr. Potter found that Claimant suffered from simple CWP based on his 14 years of treating Claimant, his review of the chest x-rays, PFTs, and ABGs. He also found that Claimant's smoking and coal dust exposure contributed to Claimant's pulmonary disability. Moreover, Dr. Potter found that Claimant had pulmonary fibrosis based on CT scan and chest x-ray evidence. Despite his lengthy treatment history with Claimant, Dr. Potter did not address the role of Claimant's obesity, coronary artery disease, or pulmonary emboli on Claimant's pulmonary capacity. Dr. James found that Claimant suffered from a diffuse interstitial lower lobe lung disease secondary to the chronic inhalation of coal mine dust that was responsible for Claimant's pulmonary function and oxygenation abnormalities. Dr. James ruled out COPD,

emphysema, bronchitis, idiopathic pulmonary fibrosis, coronary artery disease disease, obesity and Claimant's shunt as potential causes of Claimant's pulmonary abnormalities. However, Dr. James relied upon medical literature that other physicians opined failed to control for cigarette smoking. Drs. Renn, Rosenberg, and Repsher also acknowledged that cigarette smoking and GERD can lead to the development of linear opacities in the lower lung zones. Dr. James' did not address these criticisms, nor did not rule-out cigarette smoking and/or GERD as potential causes of the lower lobe linear opacities. Additionally, Dr. James ruled-out Claimant's shunt as a possible cause of his oxygen abnormalities because Claimant has no evidence of the several causes of a shunt. However, the opinions of Drs. Renn and Rosenberg identified Claimant as being at a high risk for developing pulmonary emboli, which can lead to the development of a shunt. The evidence, especially the 1995 pulmonary angiogram established a past history of pulmonary emboli.

In contrast to the two physicians who found that Claimant suffered from CWP, Drs. Farney, Rosenberg, Renn, Tuteur, and Repsher all agreed that Claimant did not have CWP. These five physicians agreed that Claimant suffered from a gas exchange abnormality and possibly a minimally insignificant degree of COPD. All five also found that Claimant had a history of recurrent pulmonary emboli that led to a right to left shunt, which caused the gas exchange abnormality. Dr. Farney opined that the most recent CT scan and chest x-ray did not show pulmonary fibrosis. He attributed his finding of a reduced diffusion capacity to Claimant's emphysema caused by cigarette smoking. Dr. Rosenberg opined that the linear opacities that appear on some of the chest x-rays were not consistent with the rounded pattern of opacities associated with CWP. Drs. Rosenberg and Renn both identified the ABG conducted while Claimant was on 100% oxygen as significant for identifying a shunt as the cause of Claimant's gas exchange abnormality, since 100% oxygen corrects all abnormalities except for those caused by a shunt. Dr. Rosenberg suggested that Claimant's history of GERD could be responsible for the chest x-ray findings of linear opacities. Dr. Renn stated that the 1997 and 2002 CT scans showed evidence of bibasilar interstitial fibrotic changes. He argued that there was no evidence of COPD, emphysema, or bronchitis. Dr. Renn also noted that Claimant was at a high risk for suffering pulmonary emboli. He attributed Claimant's reduction in diffusing capacity to Claimant's GERD and recurrent pulmonary emboli. Dr. Tuteur found that Claimant's coronary artery disease, in addition to Claimant's right to left shunt, contributed to Claimant's respiratory function impairment and gas exchange abnormalities. Similarly, Dr. Repsher linked Claimant's GERD to Claimant's reduction in diffusing capacity and the linear opacities present on the chest x-rays. He sharply criticized Dr. James' diagnosis of lower lobe fibrotic CWP.

I find that Claimant has failed to establish the presence of pneumoconiosis by a preponderance of the newly submitted narrative medical opinion evidence. The February 26, 2002 CT revealed an interstitial lower lung lobe disease that was not consistent with pneumoconiosis. Dr. Potter's opinion, while reasoned and documented, is controverted by the opinions of Drs. Farney, Rosenberg, Renn, Tuteur, and Repsher because their opinions are better supported by the objective evidence and because their conclusions are supported by more thorough rationale. Dr. James relied on suspect medical literature to attribute Claimant's lower lobe interstitial disease to coal mine dust exposure. That opinion was contradicted by the opinions and deposition testimony of Drs. Renn, Repsher, and Rosenberg. While the exact cause of Claimant's lower lobe interstitial lung disease cannot be convincingly determined from the

record, it is clear that Claimant's exposure to coal mine dust is not a significantly contributing factor to its development. The evidence establishes that linear lower lobe opacities are not consistent with a diagnosis of CWP. Additionally, the record identifies cigarette smoking and GERD as possible causes of the linear opacities. Dr. Renn's opinion establishes that Claimant does not even meet the criteria for diagnosing pulmonary fibrosis. The preponderance of the evidence also establishes that Claimant's gas exchange abnormality was caused by a right to left shunt brought about by recurrent pulmonary emboli. Claimant was a high risk candidate for developing pulmonary emboli, and he had a history of recurrent pulmonary emboli. Even if Claimant did suffer from a minimal amount of COPD, the preponderance of the evidence establishes that Claimant's smoking history was the causative factor. Moreover, even if Claimant suffered from a reduced diffusion capacity, Drs. Renn and Repsher found that Claimant's recurrent pulmonary emboli and GERD more than adequately account for any reduction. Therefore, I find that Claimant has failed to establish the presence of pneumoconiosis under subsection (a)(4).

Claimant has not established the presence of pneumoconiosis through newly submitted evidence under any applicable subsection of § 718.202(a). Therefore, I find that Claimant has not established that he suffers from pneumoconiosis.

Entitlement

Claimant, Charles Byrge, has failed to establish a change in conditions or a mistake in determination of fact in the prior denial of benefits sufficient to meet the statutory requirements of § 725.310. Mr. Byrge has not established that he is totally disabled due to pneumoconiosis arising out of coal mine employment. Therefore, I find that Mr. Byrge is not entitled to benefits under the Act.

Attorney's Fees

An award of attorney's fees is permitted only in cases in which the claimant is found to be entitled to benefits under the Act. Because benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for the representation and services rendered in pursuit of the claim.

ORDER

IT IS ORDERED that the claim of Charles Byrge for benefits under the Act is hereby DENIED.

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THOMAS F. PHALEN, JR.
Administrative Law Judge

NOTICE OF APPEAL RIGHTS

Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing notice of appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. **A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.**